

ventricular arrhythmia in acute myocardial infarction (AMI) canine model.

METHODS Sixteen dogs were randomly divided into LF-EMF group (n=8) and Sham group (n=8, with sham LF-EMF). LF-EMF (1Hz; stimulation time 8s; interstimulus interval, 5s) was delivered to the surface area of LSG for 30 minutes. Blood pressure (BP) elevation and T-peak to T-end interval (Tp-e) change in response to LSG stimulation, and LSG neural activity were measured at baseline and 30 minutes after LF-EMF. Then AMI was induced by ligating the left anterior descending coronary artery for 1 hour, and LSG neural activity and ventricular arrhythmia prevalence were recorded.

RESULTS At baseline, LSG stimulation resulted in a significant increase in BP and prolongation in Tp-e, which were significantly attenuated by 30 minutes LF-EMF in LF-EMF group and kept at a comparable level in Sham group. As compared to group baseline, 30 minutes LF-EMF resulted in a significant decrease in LSG neural activity, whereas no significant change was caused by sham LF-EMF. Furthermore, the AMI induced activation of LSG and ventricular arrhythmia was significantly suppressed in LF-EMF group when compared to Sham group.

CONCLUSIONS Noninvasive LF-EMF can suppress left stellate ganglia (LSG) neural activity, thereby reducing ventricular arrhythmia in AMI canine model.

GW26-e2472

Involvement of vascular peroxidase 1 in angiotensin II-induced eNOS uncoupling

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OBJECTIVES Endothelial nitric oxide synthase (eNOS) uncoupling contributes to a decreasing of NO level and endothelial dysfunction. Evidence has accumulated that eNOS uncoupling was induced by oxidative stress. Vascular peroxidase 1 (VPO1) is a newly identified haem-containing peroxidase that catalyzes hydrogen peroxide (H₂O₂) to generate hypochlorous acid (HOCl) and aggravates oxidative stress. The aims of this study were to determine the potential role of VPO1 in eNOS uncoupling.

METHODS Cultured human umbilical vein endothelial cells (HUVECs) were incubated with angiotensin(AngII)(10⁻⁶M) or HOCl (200uM) for indicated time. VPO1 and eNOS expression, the ratio of eNOS dimer and monomer, the H₂O₂ and HOCl levels were measured.

RESULTS Incubation of AngII (10⁻⁶M) for 24h significantly enhanced the protein and mRNA expression of VPO1, the levels of H₂O₂ and HOCl while decreased the total eNOS protein expression of and NO level. The angiotensin II-mediated eNOS uncoupling was inhibited by knockdown of VPO1 using small hairpin RNA. Furthermore, eNOS uncoupling was found in purified recombinant eNOS with the treatment of HOCl (200uM) or VPO1/ H₂O₂/HOCl system.

CONCLUSIONS VPO1 is a novel regulator of eNOS uncoupling in HUVECs via HOCl generation, which may contribute to decreasing of NO level and endothelial dysfunction.

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Lack of association between CETP I405V Polymorphism and Lipid Levels and Longevity in Guangxi Hongshui River Basin

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OBJECTIVES To investigate the I405V polymorphism of the cholesterol ester transfer protein gene (CETP) and its possible association with lipid levels and longevity in Guangxi Hongshui River Basin.

METHODS The polymorphism of CETP I405V locus was genotyped by PCR-RFLP and serum levels of total cholesterol (TC), triglyceride (TG), high density lipoprotein (HDL), low density lipoprotein (LDL) levels were measured for 523 Zhuang long-lived inhabitants (aged 90 and above, longevity group) and 498 healthy controls (aged 60-75, control group) residing in Hongshui River Basin, northwest Guangxi Province.

RESULTS The levels of TC, HDL-C and LDL-C were higher but TG and HDL-C/LDL-C ratio were lower in LG than in non-LG (P<0.001 for all). The I405V genotypic frequencies were II 34.28%, IV49.36%,

VV16.36%, and allelic were I 58.96% and V 41.04%, respectively. No significant difference in allelic and genotypic frequencies were noted between the longevity group and control group (p>0.05). There was no significantly statistic difference of every lipid profiles among CETP I405V genotypes within groups, although intergroup comparison showed that the LDL levels of all the three genotypes were greatly increased in the longevity group as compared to the control group.

CONCLUSIONS Our results show that CETP I405V polymorphism is not correlated with lipid modulation in the long-lived individuals in Hongshui Region. The higher LDL level in the oldest olds may have alternative explanations which need further investigation.

GW26-e4398

Cardioprotection of PI3K-mediated signaling is required for anti-arrhythmia effects and myocardial repair of ischemic preconditioning in infarcted pig hearts

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OBJECTIVES Although the phosphatidylinositol-3-kinase (PI3K)/Akt pathway is essential for conferring cardioprotection in response to ischemic preconditioning (IP), the role of PI3K/Akt signaling in the infarcted heart for mediating the anti-arrhythmic effects in response to IP remains unclear. We explored the involvement of PI3K/Akt in the IP-like effect of connexin 43 and proangiogenic factors with particular regard to its role in protecting against ischemia-induced arrhythmia, heart failure, and myocardial remodeling.

METHODS Groups of pigs were administered phosphate buffered saline (PBS) or LY294002 solution. Before induction of myocardial infarction (MI), pigs were grouped according to whether or not they underwent ischemic preconditioning (IP). Next, all animals underwent MI induction by ligation of the left anterior descending (LAD) coronary artery. Myocardial tissues from the pig hearts 7 days post-MI were used to assess myocardium myeloperoxidase and reaction oxygen species, infarct size, collagen content, blood vascular density, expression of Akt, connexin 43, and proangiogenic growth factors, using a spectrophotometer, histology, immunohistochemistry, real-time RT-PCR, and western blot.

RESULTS At 7 days post-MI, IP significantly reduced animal mortality and malignant ventricular arrhythmia, myocardial inflammation, infarct size, and collagen content, and improved cardiac function and remodeling; use of the PI3K inhibitorLY294002 diminished these effects. In parallel with a decline in Akt expression and phosphorylation by myocardial, LY294002 injection resulted in significant suppression of connexin 43 and proangiogenic factor expression, and a reduction of angiogenesis and collateral circulation.

CONCLUSIONS These findings demonstrate the cardioprotective effects of IP on anti-ventricular arrhythmia and myocardial repair through up-regulation of PI3K/Akt-mediated connexin 43 and growth factor signaling.

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A Study of PS gene polymorphism and soluble P-selectin in atrial fibrillation-induced thromboembolism

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OBJECTIVES Thrombolism is a severe complication of atrial fibrillation (AF), and an important cause for clinical disability and mortality, whereas mechanisms for its development in AF remains partially determined. Accumulating evidence has shown connections between thrombolism and genetic factors. Protein C is an important pathway for anticoagulative system, and alteration in the protein C concentration and activity due to genetic polymorphisms is associated with development of thrombolism.

METHODS This study investigated three SNPs (-2123C/G, Thr715Pro, -1817T/C) of PS gene belonging to protein C family using PCR method, measured sP-selectin concentration using ELISA method, and analyzed its association with non-valvular AF (NVAf) to compare potential differences in related biomarkers between 102 NVAf patients with thrombolism and 200 NVAf patients without thrombolism of Kazakh and Han population.